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THE BALLISTOCARDIOGRAPH

Earlier attempts to record the impact of the moving blood in the heart and great vessels were greatly improved when Starr announced the ballistocardiograph eleven years ago.[1] The instrument is simply a light bed suspended from above or supported from below so that it vibrates in a longitudinal (head-to-foot) direction at a frequency of 10 to 15 cycles per second when loaded with a dead weight. The vibrations are undamped, save for the damping effect introduced by the recumbent subject's body, which has been estimated at 50 per cent. The frequency, then, is about the same, 5 to 7 cycles per second, whether recorded with the Starr ballistocardiograph or directly from the body, as with Dock's instrument.[2] Vibration of the bed at its own frequency may reinforce or nullify the vibrations initiated in the vascular system. Nickerson, in an effort to avoid this resonance between subject and bed, employed a low-frequency apparatus which vibrated for the given weight of the patient at a frequency of 1.5 cycles per second.[3] These movements then are critically damped by an ingenious oil system so that any displacement of the bed is corrected in the shortest possible time without overshoot. All waves then are due to forced vibrations, and, though present, movement of the body in relation to the table is minimal. The great drawback to this instrument is the fact that respiratory movements will be significant at such a low frequency; consequently, breathing must be suspended while the tracing is taken and variations in systolic output, due to Valsalva or Mueller effects, cannot be completely avoided. The Starr school contends that his apparatus best records the vibrations of the body. Nickerson feels his instrument comes closer to measuring, undistorted, the primary vibrations arising in the cardiovascular system.

Cardiac Output

Both Starr and Nickerson have made valiant attempts to derive the systolic cardiac output from their respective curves, which, though labeled the same, differ a great deal in the timing of peaks and valleys in the cardiac cycle. Each, while he gave attractive justifications for his choice of a formula, had to conclude with an empirical constant which brought the results into conventional units. Starr took the square root of the average areas under the first footward valley (I) and the subsequent headward peak (J) and multiplied by a factor. This was originally said to yield cc/stroke, but lately Starr has given the result in terms of percentage of the average normal value for the given age and sex of the subject, and prefers to refer to *maximum cardiac force* rather than to some measure of volume.[4] Nickerson chose to measure the slope of the line connecting I and J peaks, converting this to cc/stroke by factors chosen empirically. He still gives results in terms of stroke volume, though the agreement between outputs calculated by the direct Fick method and the Nickerson ballistocardiograph has not been close.

Hamilton has best analysed the difficulties involved in recording the vibrations in the vascular system.[5] If the impact of the initial forces could be directly and accurately recorded, the systolic ejection curve might be ascertained. However, the rotation of the heart, the elasticity of the great vessels, the peripheral resistance, the movement of the body on the bed, as well as the distortions imposed by the limitations of the frequency response of both the body and the recording device with which it is coupled, all conspire to defeat such an analysis.

It is still believed that the vertical amplitude of the main (I+J) headward wave must be related to systolic ejection, both to the volume of systolic output and the acceleration of the blood during ejection. If the latter could be assumed to be within a narrow normal range, changes in amplitude might

be related to changes in cardiac output, but this is certainly not always the case. Variations in peripheral resistance and vascular elasticity also need evaluation. Starr[4] has recently stated that the amplitude (of I+J) is probably related as much to the velocity curve of systolic ejection as to the actual output. The velocity curve for systolic ejection may have greater clinical significance than the output, but it has not been demonstrated that the ballistocardiogram can differentiate the two.

Variation in Form

Starr early pointed out that low amplitudes were found with diseased hearts, but also in hypothyroidism, hypertension, convalescence from acute illness, and even in certain people presumed to be normal as far as other cardiovascular determinations were concerned. Certain of the latter developed subsequent evidence of coronary artery disease. Abnormalities in form, such as notching, or unusual configurations of the I, J and K waves, proved to be associated with arteriosclerotic heart disease in high incidence. Abnormality has further been classified in various ways: with regard to the percentage of abnormal complexes, the degree of deviation from the normal during expiration, or the inability to recognize definite complexes. These have been qualitative, until recently an index of variations in amplitude with respiration has been introduced. Post mortem control has indicated that abnormal ballistocardiograms can be found in organic heart disease in a frequency as high as 50 per cent in some conditions. It is not uncommon, however, for a younger person in heart failure or with obvious coronary artery disease to have a normal ballistic form. A more careful quantitation of variations in form is necessary before these observations can become useful.

Hamilton was the first to note the significantly unique abnormal pattern in coarctation of the aorta, and this has been confirmed and also observed in other obstructions of the aorta, though the explanation for this particular pattern is not clear. In heart failure and in arteriosclerotic heart disease without decompensation there are often abnormalities in amplitude and in the intervals between various waves. A certain percentage of aortic insufficiency cases yield abnormally large deflections, and hypertensives also present a characteristic pattern. There is no useful pattern in patients with arrhythmia or moderate dyspnea. Abnormal patterns, as they become better defined, will become of diagnostic value in certain situations, though thus far they are largely confirmatory.

Abnormalities present in heart failure are often corrected to some degree by therapy, and previously low amplitude waves increase significantly. In normal hearts there is no such change with therapy, so that serial ballistocardiograms might be used in evaluating the course of therapy in selected cases.

Summary

The ballistocardiograph is an instrument designed to respond to the movements of blood in the heart and great vessels. The engineering problem of accurately representing the systolic velocity of cardiac ejection has thus far defied adequate instrumentation, so that the resulting record can be evaluated on an empirical basis only. Whether the high- or low-frequency table becomes most desirable will depend upon which ultimately yields the most information. Ballistic amplitudes can be interpreted as output only with great reservation, though there is the lingering hope that better instrumentation and understanding may yet change this. Significantly low amplitudes are found in many conditions, including certain abnormalities of the cardiovascular system, but not necessarily those of dire prognostic import. Certainly abnormalities of the ballistocardiogram derive from abnormalities in systolic ejection and, with greater experience, will allow diagnostic and prognostic implications. Ultimately, the ballistocardiograph will probably achieve usefulness in screening prospective cardiac patients and in serial studies for better control of therapy. It will never compete with the electrocardiogram, for it measures mechanical rather than electrical phenomena of cardiac contraction. It presents no danger to the patient and merits exploration for all possible clinical value.

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